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Proceedings

of the

Second Annual American Association

of

Equine Practitioners Convention

Chicago, Illinois

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Second Annual
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Convention

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FOREWORD

This is the first proceedings book of the American Association of Equine Practitioners and covers papers presented before the Association at the Second Annual Convention held in Chicago, Illinois, December 16th and 17th, 1956. Some papers from the First Annual Convention of the Association were published in the Journal of American Veterinary Medical Association, Volume 128, Number 12, issue of June 15, 1956.

This book was prepared from papers presented or transcribed from tape.

Brigadier General W. O. Kester, President of the American Veterinary Medical Association delivered the major address at the Annual Banquet held during the Convention.

Doctor John D. Gadd, Equine Practitioner of Cockeysville, Maryland, presented excellent movies of surgery on "The Rectovaginal Fistula" and for prevention of "Cribbing" with commentary which taken alone does not lend itself to publication.

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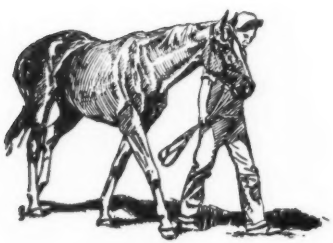

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Abstracts from Address on

FRACTURE OF THE FIBULAE

by

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The original report on this condition was published in July, 1956. Since that time a rather intensive study of the condition as it occurs in various breeds of horses has been made. Radiographic studies of consecutive patients of the equine species presented to the Stange Memorial Clinic for treatment of various conditions were made as a part of this study, and from these it was found that the fracture occurs most frequently in the Standard, and secondly in the Thorougbred. If the fracture was found in other breeds of horses, there was usually a history of some type of injury to the stifle area to correlate with the fracture. Also, it was found that the history of the lameness, and the physical symptoms exibited were typical enough to make a tentative diagnosis of the condition or at least indicate the need for radiographs to confirm the diagnosis.

The history in racing animals was usually that of a lameness following a race or a fast workout, this being followed by some improvement upon rest. If the animal was again raced or worked, the lameness would recur. In a variable length of time the acute lameness would disappear, only to be followed by rough gait, bearing in or out, or by breaking gait. Associated with this would usually be a history of a muscular soreness over the lumbar and/or the gluteal regions. Occasionally, the history would just be a complaint by the owner that the animal showed an indefinite rear leg lameness, or that the owner suspected "kidney trouble" since the animal was sore and stiff from the myositis. In certain cases there was a history of the animal being cast, or of other type of strain or trauma to the leg such as getting the leg caught in a sulky wheel, or in a manger; or some other twisting strain.

When these animals are examined clinically, it is often possible to detect a peculiar looseness or abnormal motion of the stifle joint when the animal is moved at a walk. Very rarely, it is possible to elicit pain upon palpation of the stifle area. Pain is commonly produced by deep palpation over the lumbar and/or gluteal regions. This is due to the myositis.

Radiographic study of the stifle will confirm the diagnosis. The fracture may be single, or there may be a double fracture of the bone. The most usual site of the fracture is about 1½" below the head of the fibula; in case the fracture is double, the second fracture is usually from 1" to 1½" below the first. In a small percentage of cases, the fibula will be found to have developed incompletely. In these the head of the fibula will be present but the midportion of the shaft, or the entire shaft will be absent, being replaced by a band of fibrous tissue. These cases are not considered to be abnormal.

Radiographs will also show that in most of the younger animals in which lameness is present, that the tibial tuberosity shows evidence of an abnormal pull by the patellar liagaments. This pull may be so severe that the tuberosity is acutually torn loose at the lower part of the epiphyseal junction with the tibia. Care should be taken in the interpretation of this, as the epiphyseal junction between the tibia proper and the tibial tuberosity is not completely ossified until about the age of 4 years. In older animals the pulled tibial tuberosity will show on radiographs as an abnormal enlargement or an abnormal angulation of the tuberosity.

Our theory as to the cause of the lameness produced by the fracture of the fibula is as follows: The fibula itself is not a weight-bearing bone, so that the fracture in itself cannot cause a supporting lameness. The lateral ligament of the stifle has its main attachment to the head of the fibula. When the fracture occurs, the site of the fracture is just below the attachment of the ligament. This indicates that the lateral ligament by its pull has caused the fracture, and/or that the interosseous ligament between the fibula and the tibia has been torn loose or weakened. When the fracture occurs it allows the lateral ligament to pull the head of the fibula outward, and at the same time allows the lateral ligament to relax to some extent. In turn, this allows the stifle joint to bow outwardly, as the medial ligament of the stifle is still under normal tension. When the stifle bows outwardly an abnormal amount of tension is placed on the patellar ligaments, often producing a "pulled tibial tuberosity" or even a so-called "pulled fracture" of the tibial tuberosity. In some cases there is damage to the menisci of the stifle joint, especially the medial. Since the stifle joint has abnormal motion, the muscles of the entire leg and of the back are placed under strain when the animal is moving at high speed, and this is probably the cause of the myositis that is often seen.

The treatment advised is mostly symptomatic. Stall rest for several months, plus feeding of a good mineral and vitamin supplement is recommended in the case of younger animals, especially in those in which the fracture is felt to be quite

recent. The painful muscles are often injected with cortisone, a long acting procaine preparation, or an "internal blister". The area about the site of the fracture is usually injected with a counterirritant to attempt to speed up healing (if it should occur) and to attempt to tie down or to strengthen the lateral ligament of the stifle.

In older animals the treatment is more varied. If the animal is in racing condition, but is quite sore or rough gaited, injections of a cortisone preparation into the areas of myositis and occasionally about the site of the fracture will often releive the symptoms sufficiently that the animal may be raced. Very often in these animals a rest of 10 days is advised. At other times injections of a counterirritant into the stifle area and into the areas of myositis plus stall rest for 10 days seems to be beneficial.

Animals that are to be "laid up" are usually treated by injections of a counterirritant into the lateral stifle area and into the areas of myositis. Proper nutrition and stall rest, plus re-injection of the areas in about 3 weeks are of some benefit in these cases. Occasionally, additional beneficial results are obtained by a medium blister applied over the stifle area, particiularly over the tibial tuberosity and the lateral side of the joint.

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CALCIUM METABOLISM IN EQUIDAE

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I was supposed to be one of you. Somplace along the way I was sidetracked. But I have never lost my love of the horse or the practitioner and particuraliy the combination of practicing with horses. I have been very fortunate along the way in that I have had some wonderful teachers. I went to Kentucky during my high school days and spent summers there. I spent a summer with a trainer of saddle horses at one time. The man was a very, very fine trainer, and I might say a trainer's trainer. Having spent some time with him, I asked him, "Is this all there is to it, there must be some secret to this trade. There must be a reason why you train so well and then other trainers come and buy your stock. "All I have seen here," I said, "You could do in the back yard." And he said, "That's true. All that I have told you is all there is too it. All that we do here every day, that's all there is. There are no secrets, it is just how you do it." I think now, that everyone here has been in practice long enough to know that is just about the way it is in our own profession. There can be no secrets in this profession. There must be just skillful application of what we know. Therefore we are limited by two things; one, knowledge and two, application.

I might say to start with that I didn't choose the subject. They just say, come if you will and talk about this. I haven't anything new that I know if to report since the last time I talked to a veterinary group on calcium metabolism in the horse. I might sum up what is known about this by saying there is a review on nutrition dated about 1947 in which it reviews the leading articles on nutrition of domestic animals. This review has 175 references and one reference to the horse. My offices are with the chairman of the board of the national research committee on animal nutrition, and I talk with him daily, when he's at home and I'm at home; at the same time I asked him the other day how he was able to write a report on the nutrition of a horse when all I could find was one reference in scientific literature concerning calcium nutrition in the horse. He said, "very easy, we extrapulate it all from the cow." I guess this is quite easy but is it acceptable? I am not so sure and I know that you have your doubts too. That is the situation as it stands today.

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You know in Sission & Grossman it says that all bone is made of ash & gelation and the bone is about 60/40 in that proportion. However, we all know this isn't going to hold true in all animals. This differs with age, differs with even the species or breeds of the species. It is quite common though to say that bone is about 26% ash, and of that ash about 90% of it calcium or phosphrous. Calcium is by far the heaviest constituent of the bone. Calcium is not only necessary for the bone as we all know; it is useful in the contraction of muscle fibre, and in clotting blood. But you can see since 99% of the calcium in the body is either in the bone or in the teeth that the rest of it is truly a tracer job.

There are controlling factors in calcium metabolism. The first controlling factor of course starts right with the plant. As we assumulate calcium, we take for example a given amount of calcium in grass, alfaha or anything else. That calcium to start with isn't even measured as calcium, it is measured as available calcium. Even the calcium content in the soil is given as available calcium not total calcium. This is reasonable because it doesn't matter how much is there, it is how much is there that can be used. Likewise when we come to the plant we find that it doesn't really matter much how much is there, it's how much is there that can be used by the animal. We assume for example that alfalfa, since it is one of our best sources of calcium, provides all that is necessary without any relationship to other factors involved. On the other hand to take an extreme we know that spinach although very heavy in calcium ties up practically 100% of the calcium that is in the digestive tract at the time. I believe rhubarb is another one if you want to eat the leaves; but understand that everybody doesn't want to eat the leaves of the rhubarb. Of course the controlling factor here is oxalic acid. It's a natural complexing agent in this case.

In addition to the substance itself, there are other plant substances such as we find in cereal, phytone for example. This is in the bran mash that we feed. I am doing the very same thing that everybody else has done. I am "extrepulating," because we have no data on the horse. We don't know whether this is true in the horse or not, but in test animals wheat cereal, particularly bran, even oats to some extent, has phytone which is present in the form of phytic acid that complexes the calcium and makes it unavaliable to the animal. Therefore it is another thing that the animal may have all the calcium that is necessary and still feeding these other things makes this calcium unavailable and therefore he can't utilize it.

Next come the chemical substances. Daily we are getting more complex chemical substances to use in various means. Some we can utilize to good advantages. For example, some as versene, a complexing agent used in tying up lead or depleting

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bone of heavy elements. These will also complex and deplete the body of calcium too. It's good to know this when you are, for example, using the complexing agents in one way, it is good to know the side effects that will occur at the same time.

Then of course we have the animal substances themselves like the parathyroid extracts. The removal of the parathyroids will drop the blood level immediately in calcium. This might not deplete the animal however; the animal may keep a reserve but certainly his blood level will drop immediately.

In the picture now we have so much available calcium in the animal. We've given him this we will say—now what happens when it gets in? There are a number of things that affect this calcium pickup. The first is the health of the animal. The metabloic rate of the animal if good will be very favorable; we know that, and I just mentioned it in passing. The next thing would be the amount of the substance present. Now this has a limiting factor. If you give calcium at the same time as potassium and phosphous they may tie up all of the calcium present or available. You can give it to them but tie it up again in the gut through a regular physicological reaction and visa versa by giving an excess of calcium we can tie up other of the ions present.

In addition to that the pH of the gut evidently controls the absorption of calcium. Of course this makes quite a difference in the horse and the cow, the subject from which they extractated the data. The more acid the condition of the gut, the better the absorption of calcium. The acid condition of course starts in the stomach, and as we move along the tract the condition becomes more alkaline.

There are some other variables; the amount of protein in the feed. Of course, this you might say materially affects the metabolism; it does. This may be the way it acts, but at least without a good protien supplement, we don't have a good calcium up take. However there are certain proteins or protein derivites like lyine that will by its addition double the amount of calcium pickup from the gut of the animal. There is one substance that has been reported that is even more effective in this matter and that is lactose which will more than double the calcium pick up in the gut. Again one wonders whether the lactose makes the conditions favorable or whether there is something in the lactose itself that chemically does this.

This demonstrates why for example a young cow or a bunch of pigs given mother's milk and fed calcium will pickup much more than the twin of the litter mate when fed calcium and deprived of the mother's milk, given an artificial milk for example. The calcium pickup is much greater when they have mother's milk. The factor is two fold: lycine & lactose.

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I might say here that blood is not an indicator of the calcium balance. We can not measure the calcium in the blood and tell if the animal has sufficient calcium in his diet or not. We can't even tell if this animal is in true calcium balance because in the first work that was done in this by Kenton and Holtworth in the Philippines in which they showed osteomalacia being caused by an inbalance of calcium and phosphorus. In this case they said a 1:1 or at the most 2:1 calcium over phosphorus is ideal for the horse. In showing this they tabulated their blood studies and if you look back you will find the blood of the horses that had osteomalacia had in the vicinity of 10.5 milligrams per milliliter with a plus or minus of 2 or more milligrams per milliliter. Whereas the normal animals had 11.5 milligrams per milliliter with a higher standard deviation. Therefore a single blood test would not have differenciated between the horses that were sound and those that had osteomalacia.

We have a great many more variations due to the techniques in laboratories than we have due to any condition of feeding deficiences. You can't send samples today any place and get a repeatable sample if you change the laboratory. They don't even agree on which is the best method. I say from experience, technicians or veterinarians just are not that good when dealing in these microamounts. However there are tests that are being developed very rapidly that might give us a better indication.

The true digestibility of calcium in the horse is great. I would say in some horses, particurally in those without calcium supplement it comes close to being 100 percent. This amazed me and I am sure that it will you, that a horse given calcium by mouth will take up nearly 100% of that calcium. Of course I know you have to consider its limitations. If you give him far beyond what he can use something else happens. But given the usual amount 3, 4, or 5, up to 10, sometimes the 20 milligrams which he takes daily, he will take up most of it; nearly 100% under some circumstances. He takes it up rapidly. Within six hours after he has been fed he has nearly all that he is going to take up, whereas the cow will take nearly a day to do the same thing. The diet doesn't vary this too much. In the case in the Philippines; a condition that lasted year after year then you would find the difference. Ten days on a real calcium free diet or ten days on a diet when they are practically given half and half calcium and oats; nothing happens in either the blood or the pickup. They don't get any more. How they balance this is through the (1) preferential absorption and (2) excretion. The take up, as I said is near 100% calcium. Whereas the mature animal doesn't use this calcium and what happens—he merely replaces the calcium he has. This is a big dynamic thing. It goes on all the time. He is continually replacing the

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calcium that is within his bones for the calcium that is in his feed. Now if he goes without calcium for ten days or more the same exchange keeps going on. They reutilize the endogenous calcium. The calcium goes into the blood in the same manner, goes into the gut in the same manner, but now the calcium that went into the gut yesterday goes back into the stream again today. This keeps going and it's a very, very fluid, very dynamic thing. It doesn't stay fixed as we're apt to kind of theorize on this. It is as I said very dynamic and very continually changing. Now we note that in this exchange and here, I should point out, that there are two ways it gets into the bone, (1) by the growth, which we will demonstrate a litter later on in the pictures and (2) by exchange. I am speaking about exchange particurally now. This exchange in is molecule for molecule. However since 90% of the calcium is in the bone in the first place, the exchange in is very rapid. The chance of that getting out now is less than 99 to 1. Chances that it will get back out again are very much less because there are a whole lot of other molecules that must get replaced before the other molecule can come out. It's a matter of just chance, but in one case you have millions of molecules against thousands. However I think you get the idea that the exchange into the bone is very rapid, the exchange out of the bone is slow. Therefore, if anything gets into the bone in place of the calcium, this is the area in which the fastest uptake will occur. If for example some other substance is put into this and there are; the homologues can be taken from the second group on the perodic table, any of these will move in with about the same rapidity as calcium. If anything is given that takes the place of the calcium it's going to be there for some time because it has got to wait its turn out again and this will weaken the bone. We do this, the one that I'm working on, the homologue that I hear the most on today, of course, is strontium, and we are working on that. Not because of the harm it is going to do physically because there's too little of it, but because of the harm it is going to do radiologically. They go in as homologs but they don't go out as fast as they go in.

The calcium in the horse unlike the dog or the human is excreted principally in the feces. The urine has very little calcium in it. If for any reason calcium has begun to be excreted in the horse then we are close to pathological conditions; something has interfered with the normal metabolism of the calcium when this occurs.

Another thing that is very queer and hard to understand is why an animal that has been fed sufficient calcium goes into a negative calcium balance; that is it excretes more calcium than it takes in, at the same time it needs this calcium. It seems to be a physological thing that occurs in early pregancy. For example the mare, if it is like the cow, and we have no assurance

it is, will during the time of heavy lactation take the calcium from its bone. Now there isn't anyway that you are going to stop that by feeding more calcium because it will excrete the additional calcium. It just will take it from the bone unless we find out why, and we don't know today the why of this negative calcium balance. We don't know how we can get around that, how we can keep this animal from depleting itself. The same is true of an injured animal. We can take a horse with a broken leg and we wonder what happens to the other one. They assuredly will deplete themselves of calcium in spite of the fact that they are being fed all of the calcium that they need. Now, this doesn't mean that there isn't any answer, it means we don't know the answer. We don't know what to do in this case. We can't know until we find out why this is so. The why of the negative calcium balance.

I have some pictures here, I apologize to those who are here who have seen them before, but I will show them now for the benefit of those who haven't seen them.

PRINCIPLES OF PARASITE CONTROL IN PRACTICE

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It is customary to define a parasitic infection as one which is characteristically chronic and insidious. Clinical signs of parasitic infection ordinarily are a direct expression of the number of parasites present within a host animal. This is to say that without massive exposure and massive infection it is difficult to measure effects of parasitic infection with our ordinary clinical criteria.

For the purposes of production in livestock it is perhaps easier to define parasitic infection as characteristic "herd infection." The infections exist within the members of the herd but become noticeable when the exceptional animal becomes overwhelmed so that we may say clinical parasitism represents a movement of herd infection into an individual animal.

Probably only two of the parasitic roundworms cause a clinical infection in equines with any frequency. These two roundworms would be the bloodworm and the white worm or Parascaris.

The universality of infection in equines is widely understood and accepted. Less clearly understood is the enormous number of roundworm parasites which can be found in the digestive tract of even the healthiest animals. While the exact effects of all of the species of roundworms upon the horse have not been elaborated it can perhaps be said that there is agreement that at least they are not "good for horses."

By custom the bases for the prevention and control of infectious diseases are sanitation and management. There is a growing appreciation that standards of sanitation and management cannot be high enough on a practical basis to prevent parasitic infection or, indeed, even to minimize it. It is known that even at the best horse farms that worm parasitism is universal where dependence is placed solely upon sanitation and management to prevent or to control infection.

Moreover, worm parasitism can be covered up by improvement in diet. In fact, improvement in diet is an old and established method of covering up infections of all kinds in any livestock production.

From the viewpoint of the research parasitologist the primary barriers to infection, sanitation, management and feed-

ing, must have added to them the practice of preventive treatment. Preventive treatment realistically means treating one animal to prevent survival of infection and exposure of another animal.

Whenever a basic science is moved into veterinary practice the basic science generally suffers from its over-abundance of information. The pure sciences which deal with infectious diseases, among which parasitologists include the worm parasites, universally are based upon detailed taxonomy. Remembrance of taxonomy in basic training is known to inhibit employment of a pure science in practice. The practice of parasitology is not related to detailed taxonomy. The practitioner need only be able to recognize the broad groupings of parasites and their location within the host animal.

Parasitology also has suffered in practice because in times past arbitrary standards were established for levels of "important" parasitism. This is to say that in training the veterinarian is taught that a certain worm egg count must be found in the diagnosis before the infection can be considered of such a level that treatment is necessary. *If preventive treament is to be used in equine practice the treatment must be based upon a qualitative diagnosis within the herd and not upon a diagnosis in an individual animal.*

The principles of attack upon a parasite require a restatement of the purposes of life itself. The parasite must, as its host animal must, "get enough to eat, keep from being eaten, and reproduce its own kind." If we treat the source of infection for younger animals then we remove the principal part of exposure to infection to the younger animals. The ideal anthelmintic, therefore, would be one which would destroy the parasites' capacity to reproduce without necessarily destroying the parasites within the host.

If we consider the grouping of roundworms customarily called strongyles then we know we can identify the source of infection for foals and yearlings as concurrent infection in the breeding stock. This fact was established in the surveys conducted at the Department of Animal Pathology at The University of Kentucky.

Up until 1947 or 1948 treatment of strongyle infection in the breeding stock and in the young animals was on a periodic basis and full curative treatment was used. The drug of choice for periodic treatment had become phenothiazine N.F. That periodic treatment had failed to prevent losses from bloodworm infection in the foals was only too evident by the fall of 1947. Periodic treatment failed for many reasons. One reason is that the margin of safety of the full therapeutic treatment is narrow so that the treatment is not used with any frequency. The intervals between treatment were so great that the worm parasites could enter a horse and complete their life cycles (and complete

their damages upon the host animal) before treatment was given.

In 1947 the method of preventive treatment based upon a continuous low level subtherapeutic dosage of phenothiazine N.F. was developed at one of the leading horse farms in Kentucky. This system of the 2 gram daily dose was widely adopted throughout the Blue Grass area and every other important horse center in the United States and abroad in the decade between 1947 and 1957. There arose and have persisted objections to the continuous usage of low level phenothiazine N.F. as preventive treatment for strongyle infections.

The objections were rooted in the first unfortunate experiences in the early 1940's which resulted in part from overdosing in full therapeutic treatment since the exact dosage had not been established, and from certain individual horses so eccentric that they could not tolerate phenothiazine.

Despite the fact that the generation time of worm parasites is so slow, with less total frequency for mutation than occurs in the generations of bacteria or viruses, it has been widely held that prolonged usage of low level phenothiazine would result in the establishment of species of worms resistant to phenothiazine therapy. To the research parasitologist the fact that resistant species might be developed hardly is so significant as the immediate and remarkable advantages available from the initiation of the low-level therapy. Certainly the research parasitologist would expect that even better anthelmintics might in fact be developed before resistant species of worms had been created. This last event evidently has been accomplished. The principle of continuous attack against the parasites remains the primary basis of control.

The suspicion of possible effects upon the host animal were enhanced by announcements in the early 1950's of effects of the drug phenothiazine upon action of the thyroid in white rats. These first reports proved later to be premature in that failure of rat thyroid to take up marked iodine when the rat feed had been contaminated with phenothiazine N.F. was found to be related to the excess of iodine present in phenothiazine N.F. and not to the drug itself. It is a measure of the high caliber of investigation in Thoroughbred research that effects upon the thyroids in horses were so shortly disproved through the activity of the Grayson Foundation and the research at Oak Ridge, Tennessee.

The whole question of toxicity of the newer anthelmintics, including phenothiazine N.F. undoubtedly is derived from the past deadly history of anthelmintics which once consisted of such materials as lead arsenate, sodium fluoride, carbon disulfide, etc.

It may be said that objection to continuous low level therapy

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is related to the classical conception of veterinary medicine and its practitioners as men who repair damage only. The present maturity of the veterinary profession is reflected by its present emphasis on preventive medicine. In contemporary veterinary research hidden infectious disease now is regarded as a condition the removal of which will increase production. Preventive medicine places the veterinary profession upon the positive side of animal production.

The source of Parascaris infection was established as the infective egg and not as concurrent infection in the breeding stock. Since none of the present anthelmintics can be administered on a continuous low level basis then preventive treatment of Parascaris must be based upon periodic treatment with full curative doses. The drug of choice in equine practice at the present time is carbon disulfide. The dosage varies from 3 to 6 drams dependent upon the size and condition of the horse. The tendency is to treat horses on full feed so that the effect of the treatment upon the individual animal will not be magnified by a previous period of starvation. In point of parasitological fact anthelmintics are more effective in animals on full feed.

The introduction of the compounds known as piperazines in 1955 gave promise of a safer ascaricide for horses.

The piperazines are primarily ascaricides. Secondarily, the piperazines have an action against strongyle parasites although in most instances the action of piperazines against strongyles does not approach the effeciency of phenothiazine. The place of piperazines in preventive anthelmintic therapy needs to be established by a detailed study. Certainly the present picture of piperazines is confused because of the great number which have been marketed. It should be remembered that the piperazines have a minimum effective dosage and they can be used in periodic full therapeutic treatment only. The history of the attempt to control parasites with periodic therapy with phenothiazine should be kept in mind as the piperazines are being appraised for equine practice.

Finally, perhaps nowhere else in livestock production is it possible to achieve the teamwork among the practicing veterinarian. the research worker, and the owner or manager that there is in equine practice. This author would be remiss if he did not again express his appreciation for the privilege of being a part of such a team at the Kentucky Experiment Station in the years 1947-1951.

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LECTURE TO AMERICAN
ASSOCIATION OF EQUINE PRACTITIONERS
1956 CONVENTION
La Salle Hotel, Chicago

Preventive Veterinary Medicine and Practical Application of Alpha Tocopherol In Horses

Lecture of J. BUCHANAN CHASSELS D.V.M. V.S.
Equine Practitioner
Springbrook Farm,
Brampton, Ontario, Canada

INTRODUCTION

Gentlemen:

I am very appreciative of the opportunity to address the Equine Practitioners of America. May I first of all apologize to you for presenting myself to you in this condition. A horse rolled on my leg causing a luxation and compound fracture of my metatarsals and as there was not a good veterinarian present I had to be content with a human doctor. Of course I do not mean that, we all owe a great deal to the medical profession and I was very fortunate in obtaining a very good surgeon, expertly trained in modern techniques, and being medically minded, I was interested in all procedures. For three weeks I was lying on my back perfectly still, with my leg elevated on a Braun splint. I will not have a walking cast for two months. These facts accentuate he difficulties under which our Veterinary Osteo-surgeons work when repairing fractures, and I think that such men as Dr. Leroy Johnson are indeed a credit to our profession.

I think we must all face the fact that our specially is comparitively dangerous work.

Fortunately I had an insurance policy which gives me a small income during my period of convalscence. I hope everyone in this room has some protection in this respect.

I have been asked to give a paper on Vitamin E or Alpha Tocopherol, but, I would first like to give you an outline of the organization where the work was done. National Stud Farm at Oshawa and Windfields Farm at Toronto are owned by Mr. E. P.

Taylor. The managing director is Mr. F. S. Darlington. On both farms we have accomodation for 275 head of horses and are usually operating at capacity. Windfields Farm could be classified as a private operation whereas at National Stud Farm, all the breeding—foaling and outside customers are taken care of. We have approximately 2000 acres.

From the beginning we have been very concious of nutrition, for in Canada, we are handicapped from a climatic standpoint in that our season for pasture and sunshine is approximately six weeks shorter than it would be in a state like Kentucky. We knew that to raise good horses we must have a sound nutritional program. We must have a strict parasite control and the best pastures possible. Then we must have a good veterinary program. All of these requisities have been taken care of. Probably the best proof of success has been that in eight years we have raised six winners of Canada's best race, the Queen's Plate.

VETERINARY PROGRAM

On the sheets which have been handed out to you is a systematic outline of the veterinary procedures carried out in one year on a breeding farm, for stallions, broodmares, foals and yearlings. If you care to, you could add race horses and/or show horses. In 1957 we will have a separate sheet for race horses.

This sheet is put out weekly by the farm secretary, who types two sheets, one for the veterinarian and one for the farm foreman. All veterinary procedures must be marked on the sheet, whether done by the foreman or the veterinarian. The names of the animals are typed under their category and the date when the administration is due is typed in the square under the treatment.

As I mentioned before, to raise and train a good horse you need:

1. A good, well-bred, sound individual to start with
2. A well balanced nutritional programme
3. Plenty of exercise (bear in mind we are raising an athlete not a show animal, therefore we must develop muscle not fat)
4. A good veterinary programme.

Incidently, before I start the veterinary programme, I would like to say that we practice 4½ to 5 month weaning, it is only common sense to me that after this time the mares milk starts to diminish. In reality, if she is in foal she is feeding three, and we have noticed that after weaning at this age and supplementing the diet, the foals do better and the mare has a better chance to build up her nutritional reserve for her next foal.

XUM

PHENOTHIAZINE

All breeding stock is on the low-level, powdered phenothiazine, two grams per day for twenty-one days and off for ten days.

LIQUID PHENOTHIAZINE

Is administered if our fecal examinations show higher than 2 x. One ounce per day for three days is given to clear up the condition plus the low level to keep strongyles under control.

WASHING STALLIONS

The genitals of stallions are washed with a solution of Dettol once a week during the off season and, of course, before and after each breeding.

BROODMARES

The udder and vulva of broodmares are washed daily for three weeks prior to foaling, and before breeding. Remember to wash with clear water after cleaning, as disinfectants may destroy sperm.

FOALS

If scouring, wash daily.

FEET

The feet are examined monthly by the veterinarian on all stock. Of course, with foals and weanlings, it is most important to do corrective shoeing or trimming in the early stages.

FECAL EXAMINATIONS

Stallions, broodmares, foals, yearlings have four fecal examinations per year, and heavily infested animals are tested more frequenty until clean.

RACING STOCK

I believe racing stock should be tested every six weeks and treated accordingly.

It is amazing how quickly an animal's test will be elevated even in a short period of time.

Bear in mind that if eggs are seen under the microscope, parasites are most certainly present. If however, eggs are not seen, it does not prove that the horse is negative for parasites. I have seen some large flocks of chickens where there were no eggs.

I am only trying to emphasize that sysematic worming must be done, even though tests show negative.

On many occasions, I have had a good fecal report and for some reason later carried out a post mortem to find many parasites present.

TEETH

On stallions and broodmares, teeth are floated twice a year usually, spring and fall. This prevents the enamel points from ever becoming sharp. Why wait until the mouth is torn and bleeding causing interference with mastication and digestion?

YEARLINGS

Their teeth are floated in the fall usually just after breaking.

RACING STOCK AND SHOW HORSES

They are also floated twice a year. Three and four year olds I think should be checked at least four times per year. A loose cap may be decaying or protruding into the lips or tongue and can cause a great deal of trouble such as

(a) Throwing the head when the rider pulls on the reins.

(b) Interfering with mastication.

(c) Pain suffering and infection to the animal.

LAYMEN DENTISTS

I am told that no good veterinarian has time to do teeth, and usually leaves that to a layman.

Certainly I for one would prefer to do any other operation, but teeth.

But, in my opinion, we must all do our duty and do this work.

I can not be convinced that any layman has the proper training of anatomy physiology and pathology to do this work properly.

Last year, a price list was handed around at this meeting. I have seen the recommendation for the charge on the teeth.

Perhaps we can make our money easier on other operations, so therefore we train ourselves further in other fields.

I would suggest we increase our fee to make it worth our while to do good dental work. If not, at least refer it to a veterinarian or graduate assistant.

JOINT ILL

Mare:—are injected with a mixed bacterin six months after pregnancy and the tenth and eleventh month thereafter.

Foals:—are administered five c.c. Combiotic immediately after foaling, and the dose is repeated the following day.

This disease has been reduced to a minimum, and I believe credit is due to the above procedures.

XUM

VIRUS ABORTION

We are using Hamster Vaccine, five c.c. starting the first of September and repated every sixty days to within two months of foaling.

I was alarmed to hear about the reported mishaps Dr. Doll has encountered with this vaccine, but, after talking to him, we are still continuing this practice. We once had an outbreak of virus abortion and we would prefer the odd case of anaphylactic shock.

We are all indebted to Dr. Doll and the wonderful research he is doing.

CARBON DISULFIDE AND PIPSOL

Carbon Disulfide—Stallions, broodmares, yearlings, racing stock and show horses are administered Carbon Disulfide by way of the stomach tube around the fifteenth of November and again in February. Of ccurse no in-foal mare is administered Carbon Disulfide within two months of foaling.

The reason for using Carbon Disulfide at this time is because of its usefulness for Bots as well as Ascarids.

Piparazine:—Sucklings are administered three drams of pipsol at eight. twelve, and sixteen weeks by way of the stomach tube.

Weanlings are dosed on October the fifteenth as required, may I point out that dosage will vary with the brand of piprazine used.

I believe in using the stomach tube, to me it seems more professional, and it administers a more accurate dose into the animal than powder on the feed or drench, much of which ends up on the floor.

PREGNANCY TEST

A. Z. Blood Test:— with rats may be done at forty-five days after breeding and, in my opinion, is fairly accurate.

Manual:—examination by the trained hand may be done at this time and its accuracy will vary with the operator.

We do a second manual in the fall simply to confirm earlier diagnoses, and to check for any abortions which might have occurred and not been noticed by the men.

Apparently many abortions occur around the ninety day period and we want to know in the fall if this has happened, so we will have a better chance to treat the case, and have the mare in good condition for breeding in the spring.

TEASING

Is carried on daily during the breeding season by men

trained in this work. Accurate charts are kept on each individual mare.

The mare is teased even after she is examined in foal, and any abnormalities are reported to the veterinarian who can use his judgment in regard to speculum examinations etc.

We do not tease after the first of August, but, I think perhaps it is a good idea to tease barren mares in the fall to insure proper diagnosis and treatment, you are all aware of the fact that swabs are best taken when the mare is in Oestrus.

BLOOD LEVEL

Routine complete blood tests and Urinalysis are taken twice yearly on the Breeding stock and three times per year on Racing stock.

To this group I need not go into the tremendous value and help in diagnosis of these tests.

When an animal is not doing well, our first procedure is to take a Blood, Urine and Fecal test.

H. I. FACTOR

In January, we take blood samples from all our broodmares and our stallions. From these samples are tested:

1. *The in-foal mares*—

The serum from the in-foal mare is tested with the R. B. cells of the stallion to which the mare was bred, by simple cross-matching. If agglutination occurs, we predict the arrival of an Icteric type foal.

It is interesting to note that in the past six years, every prediction of a positive test made by our Haematologist, Dr. Humble, proved to be an H. I. type foal.

These positive mares, of course are watched very carefully, and after the foal is born, it is not allowed to nurse its dam. Tests are made immediately with the foal's R. B. cells, and the dam's colostrum, and if they are positive (and to date they always have been) there are two ways of handling these foals; firstly, by obtaining a nurse mare as foster mother, and secondly by muzzling the foal, feeding supplement, and milking the mare until the colostrum gives a negative result. We have found that in both methods, their advantages and disadvantages balance one another, and either way is quite satisfactory.

2. *Barren and in-foal mares*:—

The blood samples are tested of those mares, which have future matings to the stallion chosen for the coming year.

Usually, if we get a positive reaction, we can persuade Mr. Taylor to change the stallion. It is not absolutely necessary, but, if no change is made, we can certainly expect an H. I.

type of foal. Of interest, is the fact that on our second examination the following January, we have had a number of negative mares change to positive. I am told that this might be caused by our Virus Vaccine of equine origin. As this is the first year that we have used Hamster Vaccine, I am wondering if there will be any changes this coming January. At any rate, it is beyond my powers to explain this reaction. Dr. Humble tells me that a mare may become immunized in subsequent pregnancies, eg. there may be no stimulation to these antibodies until subsequent pregnancies.

TETANUS TOXOID AND ANTITOXIN

1500 units of Tetanus Antitoxin is administered to foals shortly after birth, along with the combiotic.

Weanlings are given 5 cc of tetanus toxoid after weaning, and another 5 cc six weeks later.

The entire breeding and racing stock are given a booster injection of toxoid every January.

This gives approximately a year's immunity against tetanus and in my experience, this has been very successful.

ENCEPHALOMYLITIS VACCINE

Due to recent outbreaks of Encephalomylitis at race tracks this year, we are going to administer Encephalomylitis vaccine, Eastern and Western type combined, to all horses.

RABIES

In Canada last year, we had an outbreak of rabies, so all horses, both breeding and racing stock, were given rabies vaccine. To my knowledge, there was no report of a rabies case involving horses in Canada during this outbreak. We have a great many foxes in the vicinity of our paddocks, so we felt the vaccine was worthwhile.

TRIPS

This is simply a record of trips made to the farms.

INSURANCE

Renewals come up annually, and the expiration dates are recorded in order that I do not miss an examination.

FOALING DATES

These dates are figured on the basis of 340 days, which we have found to be an average on many records. Of course, each mare is individual in this respect.

FOALING

After a mare foals, the following procedure is recorded:

All our in-foal mares are watched closely by a night watchman, especially when they are close to the approximate foaling date. These mares are groomed every night, and the udder and vulva washed with a mild solution of Dettol.

When the mare shows signs of foaling (sweating and nervousness) the watchman alarms the veterinarian and extra help. Clean tailbandage is applied, and the vulva and anus washed with surgical soap and mild disinfectant.

I wear a rubber (foaling suit) and sleeve length rubber gloves, all of which have been autoclaved.

We make an early examination by manual palpation to see:

(a) If the cervix is opened

(b) If the foal is presented in normal position

(c) To determine if twins are present.

If everything is normal, we usually leave the mare pretty much alone and let nature take its course. If, however, something is abnormal it is so much easier to do obstetrical manipulations in the early stages of labor, before the mare starts to strain too much.

Caudal anaesthetic is used when necessary. Oxygen and stimulants are always nearby for emergency.

After the foal is born, the following details are checked:

MARE

Name of mare:
Owner:
In foal to:
Last breeding date:
Appriximate foaling date:
Duration of labor:
Time of foaling:
Gestation period:
Wash udder before foaling:
Tie up after-birth:
Insert uterine antiseptic
(We are now trying Furea Bolutabs)
6 c.c. Combiotic after foaling:
5 c.c. Combiotic 24 hours after:
Check temperature within one hour of foaling
Four periodic temperature checks within 48 hours
Retention of after-birth
Swab on 5th day
Result of swab

FOAL

Colour:
Markings:
Sex:
Foaling Date:
Disinfect navel stump
Enema
Periodic rectum checks for 48 hours
Watch for retained Muconium
4 c.c. Combiotic
1500 units Tetanus Antitoxin
Check temperature within one hour
Four periodic temperature checks in 48 hours
First feeding

SUMMARY OF SHEET

On the back of the sheet, the veterinarian's report is recorded under the headings of Date, Name, Diagnosis, Treatment.

This information is recorded by the secretary on an individual card for each horse, which is filed alphabetically.

This system is used out at Mr. Taylor's farms, and I have a similar system for my private practice. My secretary keeps the above records and even with small owners, we notify them when their various treatments are due. The owners and managers seem to appreciate this service, and I have a full veterinary record on each animal.

ALPHA TOCOPHEROL OR VITAMIN E

I have been asked to say something about our work on Alpha Tocopherol.

We were attracted to the work on Vitamin E through several basic factors, the first being the success in humans. Secondly, the low content of pure Alpha Tocopherol in wheat germ oil. Vitamin E was not new to the veterinary profession, but, it appeared that the dosage was entirely wrong to get best results. There had been considerable work done in small animals, cattle, sheep and hogs. The research work in regard to E content in feeds had shown the enormous quick deterioration through manufacturing and storage. Actually, it was a part of our nutritional program to insure our horses of an adequate diet at all times. We had found that proper feeding methods meant having animals working at optimum and many of the everyday problems in breeding were eliminated.

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This year, in conjunction with the breeding paper we reported on horses racing. We cannot do that as yet, since our racing does not finish until the middle of November. However, I must say that I prefer the breeding end of it since there are so many things that are factual.

There is more sound basis for comparison. I might say though, that our racing stable has had its best year to date in 1956. The problem in presenting work is made difficult due to the variety of opinions of owners and trainers. If a horse performs much better on Vitamin E therapy even for a two year period as compared to two previous years you are liable to be told that ("he's just got good") or was always cut out to be that kind of a horse or maybe the trainer found his hole card. I heard of a case where the trainer of a very large stable put half of them on Vitamin E for two months. At the end of this time he was raising the roof. Nothing had happened The entire stable had not won a race at a particular meeting. He had an excuse for one part of his horses, but, he neglected to say what happened to the other half! It did not happen in Canada or in the United States. For now we will stick to our breeding experiences.

Mr. Darlington and myself have always emphasized the time element, and patience. We have cautioned that it does not fall into the category of a *wonder drug*, and that it is strictly one of the essential vitamins in the body of humans and animals. It is not a cure-all. There is a definite place for it in veterinary practice if properly administered. For instance a horse infested with strongyles will not be helped by Vitamin E. A proper diagnosis of the individual case *must* be made. If blood, urine and a fecal analysis show negative, Alpha Tocopheral might help. I am convinced of its value in nervous, high-strung horses at the race track, and poor feeders. It is especially good in the cases of what horsemen refer to as "tying up."

AZOTURIA

Its value to stallions that are shy or impotent seems to be good. I suspect that some of our race horses who are front runners and then die in the stretch may be suffering from cardiac disorders. Perhaps Vitamin E would be of value in these cases.

In mares which have difficulty in conception—(may I emphasize here the importance of proper diagnosis; there must be present no windsucking, no infection, no tipped vulva, no mal-cervical positions)—our records indicate Alpha Tocopherol has some value in getting these mares in foal.

MEDICATIONS

For certain types of wounds, the varous Vitamin E sprays

and ointments appear superior to other medications, especially in ulcerated wounds.

DOSAGE

In horses, our experiences show that large dosages are indicated for required results.

We have given as little as 200 I.U. per day and as high as 25,000 I.U. daily. I believe an optimum dose for a horse is 3,000 per day, and a maintenance dose is 1,000 per day. It is best to sprinkle powder on animals food once daily.

NEW PAPER

If any of you are particularly interested in this work, we would be pleased to give you upon request, the results of our most recent paper. Write to me care of the National Stud Farm, Oshawa, Ontario, Canada. If others take up the work, it will help us to reach our ultimate goal so much sooner. We are willing to be of help to anybody if our past experience is of value to them.

I do not want to bore you with case histories and statistics but a summary of our 1956 paper is as follows:

The regulation of dosage to suit individual needs is an important factor. It is chiefly for this reason we have presented detailed case histories of 21 mares where an increased dosage was indicated. The other mares were on a 1000 unit daily dosage continually.

We used higher dosage levels in the following types of problems:

(a) Maiden mares. In the last two years of the experiment we have bred twelve and got twelve in foal. None of these had been off the race track for more than five months, and some of them came directly out of training.

(b) Barren mares in the spring of the year in order to regulate oestrus.

(c) Foaling mares with a history of being difficult to conceive or showing irregular cycles.

(d) All mares that give a past record of needing speculum examination in order to detect oestrus.

(e) Mares that had never received Vitamin E. (this is empirical).

The thousand unit dosage of alpha tocopherol succinate appears to be sufficient for most normal broodmares. There will be certain horses where the requirements are higher, especially in the spring of the year. We had kept 29 broodmares from our original experiment on it for one full year, and only 6 of

these required such an increased dose. We recommened year-round therapy: it is more effective.

To summarize:

Barren mares on Vitamin E	18	In Foal 16	88%
Maiden mares on Vitamin E	6	In Foal 6	100%
Foaling mares on Vitamin E	31	In Foal 26	84%
Total mares on Vitamin E	**55**	**In Foal 48**	**87%**

Throughout 1955 and 1956 we worked with 41 barren and maiden mares and got 36 in foal or 88 per cent. In the six years prior to 1954 we had worked with 57 barren and maiden mares and had 49 in foal or 63 per cent.

According to Mr. Estes and the Jockey Club statistics the percentage of registered foals on the American continent is 52 per cent. In our operations over a six year period just prior to the beginning of the Vitamin E study we had 63 per cent of foals from our mares. From our test group of 34 mares bred in 1955, *however including ten at an average age of 16 years*, we had 74 per cent, an increase of 11 per cent.

In all the subjects I have covered today, I speak only as a practical practitioner in the field. As you know, each subject is a specialty in itself, and I am sure there are specialists in each field who are far more qualified than I. I believe that we are all fortunate in having with us one of the pioneers in the field of medicine on Vitamin E. Dr. Evan Shute has worked from the human side for 18 years. There is nobody that I know of, better qualified to answer any questions that might come to your minds as the result of the paper that I have just given. I would suggest that you refrain from questions until after you have heard from Dr. Shute, at which time we will be peased to answer together.

Thank you,

*ALPHA TOCOPHEROL IN THE GENERAL FIELD OF FERTILITY AND REPRODUCTION

by

EVAN SHUTE, B. A., M. B., F. R. C. S. (C)
London—Canada

INTRODUCTION

I assume you know much more about women than I do about horses. It is really presumptuous of me to address you at all. It is possible for me to learn about horses; I know you will never master the other subject. That is what impels me to talk for a little while about women and about human fertility in general to an asscciation of distinguished horsemen.

HISTORICAL NOTE

The study of the reproductive organs in human medicine is only a few centuries old. Galen, Oribasius and others had written on this subject in Roman days, but much if what they wrote was merely fanciful nonsense. In the mid-16th century Eustachio advised a husband to push semen into the uterus of his sterile wife with his finger—and the wife conceived (1). In 1799 John Hunter did an artificial insemnation, as did Marion Sims in 1866. Brown-Sequard in 1889 at age 72 described his own rejuvenation with a testicular extract. Morris did ovarian grafts in 1895. In 1903 Ludwig Fraehkel discovered the function of the corpus luteum and thereafter the doctrine of the "primacy of the ovum" was developed. Rubin first insufflated the Fallopian tubes in the female in 1920 and Huhner's test done on fresh sperm in the vagina was described in 1921.

But Gynaecology and Obstetrics for many years concerned itself more with the hazards of surgery on the female organs and the risks of childbirth than with such problems of fertility. Indeed, when gynaecologists first turned attention to the latter it was usually to develop contraceptive techniques. "Mrs. Margaret Sanger was the first to lend financial support to a project that was concerned with an investigation of childless couples," says Hotchkiss (2). During the same epochs Urology concerned itself more with venereal and other infections, with calculi and prostatic hypertrophy than with semen. However Dr. Howard Jeck set up a Male Sterility Clinic at the New York Hospital in 1932.

*Read before the American Association of Equine Practitioners, Chicago, December 18, 1956.

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It can be seen that any intensive study of fertility in the human is really but a few decades old. Research in this general field has been greatly encouraged and intensified by the formation of the American, British, Canadian and International Societies for the Study of Fertility and Sterility—but they date back only 12 to 3 years. There have been but two International Congresses held on this subject, the first one being in 1953 in New York City.

We are dealing with a young subject, therefore, and great gaps exist in our knowledge of the fertility of both the human and of domestic and wild animals. I became interested in this aspect of my medical specialty in 1933 when I began studies on vitamin E, called the "fertility vitamin" then. In the 23 years since we have learned more about its therapeutic values, and it is to studies on this vitamin that I would now direct your attention.

GENERAL PROPERTIES OF ALPHA TOCOPHEROL

There are six principal mechanisms by which this agent, vitamin E or, more properly, alpha tocopherol can influence pregnancy. It is the most effective anti-oxidant amongst the known food factors. It conserves oxygen in the tissues, enabling them to survive on less (3) (4). Thus it is somewhat equivalent to fastening an aqua-lung on a woman when you give her vitamin E. Now remember that the foetus in the womb lives in a state of severe oxygen deficiency by extra-uterine standards. Indeed this becomes so marked at term that it exactly corresponds to the anoxia experienced on a climb to a height ¾ of a mile higher than the top of Mount Everest (5). Every unborn babe is virtually higher than the top of Everest. You can imagine what that oxygen tank on his back, vitamin E, can do to help him survive the rigors of labour and his sudden expulsion into the bright new world where oxygen and pari-mutuals are a good deal easier to find.

Vitamin E is a vasodilator, at least of the termial twigs of the arterial tree (6). We all exist with more lung, more liver, more kidney, more brain and more blood vessels than we are ever apt to use. But if we lose some of these organs by disease or accidents or the knife reserves are there for the emergency. Now the infant in the womb sits on a cushion of blood vessels, the afterbirth. Through it he derives all his nutrition from his mother, all his oxygen, all his tissue-building elements, his bones, muscles and fats, and to it he returns most of his waste products, minimal as they are. That vascular cusion is often damaged. Its vessels close off or became plugged. Large dead areas we call infarcts develop in it for reasons unknown, but probably related to local blood clotting or vessel deterioration. Alpha tocopherol helps maintain the integrity of vessel walls,

and helps prevent clotting inside the vessels. It is also concerned with the elasticity of those walls (7). It must herefore be a vital factor in maintaining optimal function of that vascular cushion.

We should look upon that afterbirth as of fundamental importance to the embryo. Just as a good foundation is absolutely necessary for a good house, so the placenta plays a major role in determining what kind of new body will develop upon it as the child grows in the womb. It has always been held that the prime duty of the obstetrician is to deliver the child at term. I suggest that this is often too easy a role to challenge his powers. Many a nurse could do as well, many a midwife,—and often does when he arrives too late. His real role, and one that often demands a good deal more skill and medical knowledge, is to care for that afterbirth throughout pregnancy so that the child and uterus ripen completely and concurrently, and are both normal when and until the time comes for them to part. Hence the role that vitamin E plays in the preservation of the placental blood vessels is of vital importance in any pregnancy.

I could add that alpha tocopherol is a fibrinolytic substance and this, too, tends to minimize the formation of the dead tissue enwrapped in fibrin which we call infarcts and which often replace much functional placental tissue (8).

There are many other functions of this polyvalent vitamin which play an important role in obstetrics. Only one of these will I mention—its ability to improve placental "cling." The afterbirth is apt to be too loosely attached to the wall of the womb. Perhaps this is related to an excess of oestrogens in the blood (9). In any case, the placental villi or fingers seem to dig into that wall more deeply when vitamin E is given, and thus many of the haemorrhagic accidents of pregnancy associated with a loosening placenta can be avoided (10). More of this later.

PRACTICAL ASPECTS IN FERTILITY AND OBSTETRICS

The writer reviewed his experience with vitamin E in Obstetrics (11) and Gynaecology (12) for the Third International Congress on Vitamin E, held in Venice, Italy, in September 1955. Only a brief summary of his views need be considered here, and those mainly of a sort that apply to the difficulties commonly encountered by horse breeders.

Fertility in the male, perhaps in the female, may be improved by vitamin E. The evidence for this in the literature is very conflicting and inconclusive, and I believe rests most firmly on work we ourselves have done. Sperm analyses are so variable, so crude, and so extremely unsatisfactory in general that studies based upon gross morphological changes in sperm spreads can have very slight evidential value. The electron micro-

scope, for example, shows us that many sperms we would have called normal are quite abnormal (13). In any case, when did the morphology of sperms become the sole arbiter of their capacity to fertilize ova? A glance at sperms does not tell what they can do. I believe that few laboratory tests are as poor as sprem studies. We should revert to a much more primitive point of view and recall that the object of sperms is to produce progeny, not to wriggle across a glass slide. It should be remembered that the decision as to whether or not vitamin E plays a role in animal fertility has often been based on the crudest sperm examinations. Sperm counts are helpful in prognosis. For example, they should exceed 20,000,000 per cc. But counts are very inconsistent, too. What matters is evidence that vitamin E can improve sperms qualitatively. The best demonstration of that is drawn from our experiences with sires of anomalous infants. If these fathers are treated with vitamin E before further conceptions the chances of normal offspring ensuing are enhanced very greatly. For example, we have succeeded in 17 out of 19 such trials. The chances of this result occurring by mere chance are smaller than 1 in 6000 (14). There is good reason, therefore, to use alpha tocopherol to improve sperm quality and fertility.

The evidence that vitamin E improves the fertility of the ovum is very far from conclusive, (12) however, perhaps the best being the observations Darlington and Chassels (15) reported last year—and they were not too extensive.

Once a pregnancy has occurred our next interest is to preserve the gestation. Too many pregnancies end as abortions, miscarriages or premature deliveries, often accompanied by debilitating or even by fatal haemorrhage and shock. What has vitamin E to offer here?

It seems to be very helpful. Indeed, a large literature has developed to emphasize this. Recently there has been a good deal if doubt expressd as to its value in preventing human abortion, since many workers have claimed a far higher precentage of success than was ever hoped for before with such simple measures as bed rest only. Others have regarded abortion as Nature's effort to purge the family tree of wormy fruit. Practical experience would make one a little skeptical of both these views however. Threatening abortions left to themselves do not do as those treated with vitamin E. Abortions that are prevented only rarely go on to produce defective infants at term. Our personal experience has been summarized elsewhere (11). It is, roughly, that we find this substance so useful in preventing premature birth (16) (17) that it often does so even after the foetal membranes have ruptured (18), something that rarely could be anticipated (19). I have no idea what the wastage from premature delivery in horses is, but we have estimated that had Canada during the last war salvaged all its premature babies

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it would have suffered no population loss from that conflict. The future of this problem surely lies in prevention, rather than in tedious, difficult and expensive efforts to salvage these prematures once they have been delivered.

Many of these premature deliveries, if not most, are associated with bleeding and occasionally this is severe enough to produce shock or even the death of the mother. A well-recognized entity, for example, is premature separation of the afterbirth, almost always associated with intrauterine or external bleeding which can reach serious proportions. We have long suggested vitamin E as a preventative for this condition and have accumulated data to indicate that by its routine use during pregnancy the incidence of this condition can be decreased by about half (11) and that the foetal salvage may be very considerable even in frank cases. This effect of Vitamin E is presumably due to its power of stabilizing the utero-placental interface and increasing placental "cling", as well as of preserving vascular integrity and keeping blood coagulability more nearly normal (20) (21). The increased foetal salvage, even in such cases as do develop during inadequate (?) vitamin E therapy, is probably related not only to vascular preservation in the placenta but to improved oxygen utilization in the precarious and intimate decidual tissues.

Let me reiterate my conviction that giving vitamin E to sires before conception goes far toward eliminating anomalous offspring. How big a factor this is among horsebreeders I have no idea—but at least it is a point to be born in mind.

LESSONS FROM VASCULAR DISEASE

Now may I turn to a little broader overview of the properties of this vitamin in vascular disease, in order to illustrate the basis of much that has been said already and perhaps to suggest further ways in which it can help you in your work.

These coloured photographs of disappearing peripheral blood clots, of capillary dilatation, of oxygen utilization in anoxic rats, of the salvage of indolent ulcers and of small gangrenous areas in the extremities, of its role in the complicated diabetic, especially of its value for burns and wounds may be as impressive to you as they have been to purely medical groups. All of these illustrate either effects that only vitamin E can produce, or effects it produces faster or more safely, leaving smaller residual defects. Other agents can heal many indolent ulcers, can heal wounds and burns, can dilate capillaries or improve the integrity of thin vessel walls—but none can do it as well or as safety as alpha tocopherol. This is widely recognized, which is why so much support has accrued to our original cardio-vascular observations of 10 years ago. For example, it seems not to be generally known that there are now 73 papers

in the literature supporting the use of vitamin E in heart disease, 27 supporting its value in thrombosis (clots within vessels), 33 for indolent or chronic ulcers, 2 for burns, 10 for capillary permeability, 17 for thromboangiitis obliterans, 26 for diabetes mellitus, 4 for fibrinolysis, 5 for incipient gangrene, 7 for kraurosis vulvae, 15 for nephritis, 7 for thrombocytopaenia, 7 for purpura, 10 for roentgenologic tissue damage, 12 for vascular dilatation and 5 for accelerated wound healing. All this adds up to formidable support, especially when it is recalled that these total more than 300 medical papers (22) and that 84 of the papers just mentioned were well "controlled" studies, such as the most exacting and critical experimenter might demand.

GENERAL ASPECTS OF ALPHA TOCOPHEROL AS A THERAPEUTIC AGENT

May I emphasize that this polyvalent vitamin, alpha tocopherol, called vitamin E in medical slang, is unique in many ways. It is remarkably safe. It is easy to take or to give even to animals. It is relatively cheap. It has been in all our diets until we "purified" them too well in recent dacades. Now we probably all are starved of it, for our average intake of *alpha* tocopherol is only 10 mgm. per day when it should be at least 3 to 12 times as much (23) (24). It has almost too many clinical applications—and yet no less could be expected of an agent able to assist bodily oxidation, where every cell breathes, and capillary circulation, where every cell needs blood, whether that cell is in the heart or the toe or the brain or the skin. Its multiple powers should never be held against it in the day of chlorpromazine, A.C.T.A., cortisone, terramycin, sympathectomy, vagotomy or lobotomy.

There has long been a belief amongest veterinarians and others that wheat germ oil was a good source of vitamin E. It is even advertised in the United States as "vitamin E"! This is grossly inaccurate. Originally wheat germ was used as a source of alpha tocopherol, but it was always known to be a very dilute source, and the oil extracted was quickly destroyed by the least trace of rancidity. Indeed, at the moment it is not even used as a commercial source by the single great firm which supplies all or almost all of the "natural" vitamin E on the world market. Soya bean oil or cottonseed oil, the sources they use, peanut oil and even margarine have as much right as wheat germ oil to be labelled "vitamin E"!

We have in our possession a letter from a great firm reporting their analysis of commercial wheat germ oil as follows:

"So-called medicinal wheat germ oil, provided it is fresh and not rancid, contains about 2 i.u. of vitamin E activity per gram. Ordinary wheat germ oil such as might be used as a component

of animal feeds may only contain half this potency. However, assuming the value of 2 i.u. per gram, the amount of wheat germ oil necessary to furnish 600 i.u. of vitamin E would be 300 grams, or about 8½ liquid ounces. This amount, a drinking glassful, of wheat germ oil would be difficult to take and would probably not be tolerated by most people for long."

So widespread has mislabelling in commercial vitamin E been in the United States that we have recently made representations again to the Food and Drug Administration about this deplorable situation.

We have now pointed out that wheat germ oil should not be labelled as "vitamin E" commercially and should not be used to dilute so-called standardized preparations of alpha tocopherol. We have further requested that the United States follow Canada's lead (1949) in *demanding* (it now "requests") that American firms in this field label their products exclusively in terms of international units (the League of Nations Standard). Finally we asked that sale of products be policed, in order that all label claims should represent content truthfully. We have a strong suspicion, derived from the 400 to 500 per cent price spread in comparable (?) American products, that this last point is of great importance.

One does not get half a result from half a dose; this is also true of insulin or penicillin. We have from the first stressed the need for adequate doeses of reliable products. Nothing less can be helpful, obviously. American therapeutic practice has long been clouded by inadequate dosage of ridiculous products masquerading as reliable "vitamin E". The National Formulary in the United States now recognizes what alpha tocopherol should be—the difficulty is that even great pharmaceutical houses may choose to ignore this. Be warned. Use only reliable products, labelled in terms of international units, not mgm. of "vitamin E".

Years ago we pointed out that it was possible to label a product quite truthfully as being 100 mgm. of vitamin E which actually contained 46 to 136 international units of alpha tocopherol (25). What this does to the price structure can readily be imagined. What it does to treatment is deplorable. No wonder the results achieved by us with reliable products have not always been capable of duplication by others using very poor or dilute preparations.

I hope that some of the human evidence here adduced may have some baring on your problems. It is always difficult to transfer results and conclusions from one species to another. However, the physiology of the reproductive and vascular systems of the horse and of man have close mammalian analogies which cannot be denied, and a survey of the one may be helpful

in the other. Veterinarians interested in alpha tocopherol have much to contribute not only to their own science but to comparative medicine, and I hope they will continue to magnify their great opportunities. Subjective evidence in the human is often confusing, but the evidence horsemen get is truly objective. After all, you cannot mesmerize a horse. What a horse study reveals is reliable evidence.

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VIRAL RHINOPNEUMONITIS AND VIRAL ARTERITIS OF HORSES*†

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This discussion presents recent findings obtained from research on viral diseases identified with the influenza-abortion disorders of horses. Attention will be given to the etiology, clinical manifestations, epizootiology, pathology, and differential characterization of two viral diseases that are manifested by respiratory catarrh, systemic illness, and abortion.

These two viral diseases apparently have been described in veterinary literature for many years as influenza, epizootic cellulitis, and pinkeye, and by many other names. It is evident, also, that respiratory and systemic disorders other than the two being considered have been described under these names. The reasons and purposes for introducing two new names have been published elsewhere[1, 2, 3, 4], and will be presented only briefly.

Abortion caused by viral infection was identified by Dimock *et al* in 1932[5] and was described in detail in 1942[6]. The disease was identified as equine virus abortion. Subsequently, this disease was identified in many countries by histopathological study of tissues from aborted fetuses. Reports by Manninger and Csontos[7] in 1941 and by Manninger[8] in 1949 associated virus abortion with a disease which they regarded as mild influenza. They learned that virus obtained from aborted fetuses caused a mild respiratory catarrh and febrile reaction in mares and other horses, and that horses were reciprocally immunized by the abortion virus and an infectious agent regarded as influenza virus.

An outbreak of respiratory disease identified as equine influenza was studied extensively at Front Royal, Virginia, prior to 1943 by Jones *et al*[9, 10]. The uncomplicated disease was relatively mild, but the outbreak was accompanied by nearly

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all complications that have been associated with equine influenza. A virus, the Army 183 strain, was isolated and maintained by propagation in young horses. Their decision to perpetuate the virus was, perhaps, the most important contribution of the investigation as it enabled subsequent identification of the Army 183 strain with the virus causing the viral abortion described by Dimock and associates.

The Army 183 strain of equine influenza virus and the abortion virus were studied comparatively by Doll *et al*[11, 12, 13, 14, 15]. When injected into equine fetuses, the Army 183 strain caused abortion, and the aborted fetuses had gross and microscopic lesions that were identical with those caused by the abortion virus. Infection of young horses by either virus immunized against the other and induced development of reciprocally specific complement-fixing and virus-neutralizing antibody. Each virus was adapted to suckling hamsters and in this host each produced identical lesions. The viruses propagated in hamsters were adapted to the chicken embryo in which each produced identical lesions. Young horses were inoculated with abortion virus recovered from aborted fetuses and their thermal, hematological, and symtomatic responses were essentially identical with the uncomplicated influenza described by Jones *et al*[10]. These findings verified identity of the abortion virus described by Dimock and associates[6] and the influenza virus described by Jones *et al*[9, 10]. Isolation and study of another virus associated with an influenza-abortion syndrome, however, required further consideration of the identification and classification of viruses causing respiratory and systemic disorders of horses and abortion by mares.

An outbreak of abortion accompanied by influenza-like symptoms was observed near Bucyrus, Ohio, in 1953[2]. A virus was isolated from two aborted fetuses. Comparative studies, employing cross protection tests, virus-neutralization and complement fixation tests, symptoms and lesions, and hematology, differentiated the newly isolated virus from the equine abortion virus[2]. The same report presented reasons for discontinuing use of the term "influenza" for respiratory diseases of horses, proposed the name *rhinopneumonitis* for the disease caused by the abortion virus or influenza virus of Jones *et al*[10], and proposed the name *viral arteritis* of horses for the disease caused by the newly isolated virus. The viruses were identified as the *equine rhinopneumonitis virus* and the Bucyrus strain of *equine arteritis virus*. Results of these studies are reviewed briefly.

Reciprocal immunity was not established following infection of young horses by either virus. Infection by the arteritis virus did not stimulate development of virus-neutralizing or complemen-fixing antibody for the abortion virus. Leukopenia caused by infecting young horses with rhinopneumonitis virus resulted

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principally from a neutropenia, whereas infection by the arteritis virus caused a panleukopenia with the lymphocytes being affected most severely. Some symptoms were common to infection by either virus. Each caused a fever and serious rhinitis. The arteritis virus caused rhintis, conjunctivitis, palpebral edema, colic, diarrhea, expiratory dyspnea accompanied by a heave line, and edma of the legs, abdomen, mammary gland, scrotum, and sheath. Gross lesions in horses infected by the arteritis virus are principally edema of subcutaneous, submucosal, and subserosal tissues and pulmonary emphysema. Microscopically, the specific lesion is a degeneration and necrosis of the media of small muscular arteries. Other lesions appear to result from damage to the arteries.

Gross lesions of rhinopneumonitis in young horses are principally rhinitis and interstitial pneumonitis. Lesions in fetuses aborted from infecton by the rhinopneumonitis virus are most constant and severe in the respiratory system. These are congestion and hemorrhage of the mucosa of the upper respiratory tract, edema of the lungs, and an excess of fluid in the pleural cavity. Microscopically, the specific lesions are intranuclear inclusion bodies in the bronchial epithelium and alveolar cells. Intranuclear inclusion bodies occur in the hepatic cells and in reticulo-endothelial cells of the lymphoid tissues, but lesions in these tissues are less frequent and are less extensive than those in the lungs. The names selected, therefore, are based on constant or specific lesions for each disease.

The term influenza has been applied to a variety of afflictions of animals and presently is used for diseases of man and swine caused by closely related viruses. The arteritis virus and rhinopneumonitis virus are not related to the influenza viruses of man and swine[1, 16], and therefore it appears undesirable to use the term influenza for the disease caused by either virus. The arteritis virus and rhinopneumonitis virus have no demonstrable relationship and produce distinctly different symptoms and lesions. It is undesirable to identify the diseases produced by the two viruses by one name (influenza). Either virus may be equally important as a cause of abortion, and for the same reason should not be identified as abortion virus. It is evident also that activity of each virus is principally the causation of respiratory and systemic infection instead of abortion. For these reasons it was proposed that use of the term "influenza" be discontinued and that respiratory diseases of horses be identified according to their etiology and specific lesions.

VIRAL RHINOPNEUMONITIS

The disease caused by the rhinopneumonitis virus and complicating or secondary bacterial infections is imperfectly understood. When associated with shipping, other stress, and

superimposed bacterial infection, viral rhinopneumonitis may be complicated with a variety of pyogenic processes of the respiratory tract as attested by the work of Jones *et al*[9] with the Army remount horses. There is very little definitive information on the role of the rhinopneumonitis virus in the influenza syndrome, epizootic cough, febrile illness, and other respiratory diseases observed in horses in training or racing stables. It is probable that filterable agents other than the rhinopneumonitis virus are involved, but extensive serological studies and isolation and characterization of infectious agents will be necessary for clarifying the etiology of respiratory diseases of horses in training or racing. Considerable information is available on the occurrence and nature of rhinopneumonitis in the breeding establishments of central Kentucky, where its principal manifestations are a rhinitis or "colds" of foals and abortion by mares.

Fetuses have been aborted following infection by the rhinopneumonitis virus each year since identification of viral abortion in 1932. Abortion has been observed during each month of the year except July and August. Very few abortions occur in September, October, November, May and June. Of 357 abortions recorded, 7 per cent occurred in December, 18 per cent in January, 30 per cent in February, 28 per cent in March, and 12 per cent in April. Ninety-five per cent of outbreaks were initiated in December, January, February, March and April, with 66 per cent starting in January, February and March. The earliest age of a fetus when aborted from naturally acquired infection is 5 months gestation. Approximately 5 per cent of 322 fetuses were aborted prior to the eighth month of gestation, whereas 13 per cent were aborted in the eighth month, 33 per cent in the ninth, 34 per cent in the tenth and 15 per cent in the eleventh. Ninety-five per cent of the abortions occurred between the beginning of the eighth month of gestation and full term. The number of abortions that may occur on an affected farm is variable and unpredictable. The average number of abortions per farm affected over a ten-year period varied from 1.6 to 6 annually. Analysis of records from 109 farms that had 250 abortions showed that 68 per cent had one abortion, 17 per cent had two abortions, and 9 per cent had three abortions, making a total of 85 per cent of affected farms having three or fewer abortions. The number of abortions on other farms has been quite variable, with 27 being the most recorded for a single farm. An abortion incidence of 20 to 80 per cent in a group of affected mares is not unusual. Outbreaks appear to occur in cycles with an interval of 3 to 4 years between periods of lowest incidence.

Incubation periods (time from exposure to abortion) under natural conditions are not known, because it is impossible to

date the time of exposure accurately. Mares may abort one to three months after rhinopneumonitis occurred in foals. Incubation periods after intranasal inoculation have varied from 40 to 90 days. Most mares abort within 15 to 30 days after intravenous inoculation, but some have carried the fetus 40 to 76 days before aborting. A pattern of abortion during an outbreak cannot be predicted. Only a few mares are known to have aborted more than once. Abortions seldom occur on the same farm in consecutive years.

Affected mares show no signs of impending abortion, no signs of ill health, and do not have a history of recent illness. Inquiry may reveal presence of respiratory disease among foals one to three months prior to abortion among the mares, but no illness was observed in the mares. The act of abortion appears and progresses rapidly. The placenta often is expelled with the fetus or soon thereafter. The mares ordinarly suffer no ill effect and the genital tract involutes as readily as after normal parturition. Mares that abort from virus infection breed as well as those that foal normally. The mare may suffer any of the accidents or complications that accompany foaling or abortion from other causes. These include dystocia, retained placenta, and tears, bruises, and lacerations of the genital tract.

A paralytic syndrome is observed infrequently in outbreaks of abortion caused by the rhinopneumonitis virus. The first evidence of its occurrence is an incoordination of the pelvic limbs, which progresses in 2 to 4 days into a paraplegia. The pathogenesis is not known, but circumstances suggest that it is related to the viral infection of the fetus. When abortion occurs or if abortion is induced during the period of incoordination or early stages of paralysis, the mare often recovers. Death of the mare is frequent when abortion is delayed.

Foals may be born alive at full term with rhinopneumonitis that was acquired prenatally. These foals live from a few hours to several days and some may survive. The principal symptoms are weakness, respiratory distress, rapid breathing, rapid heart rate and cyanosis. Treatment should include bacteriological or bacteriostatic drugs, oxygen, and supportive therapy.

Fetuses usually are living when abortions occurs, having heart action, but they seldom breathe. The chorion is normal in appearance. The amnion may be edematous and its inner surface and the foot pads of the fetus often are stained yellow from meconium deposited in the amnionic fluid.

The fetus aborted from infection by the rhinopneumonitis virus may not have any gross lesions. Approximately 90 per cent of aborted fetuses have lesions which enable diagnosis from gross examination. This percentage may be improved by micro-

scopic examination of selected tissues. Hemorrhages, although not specific, are relatively constant. The mucosal surfaces of the upper respiratory tract, mouth, and pharynx are often congested, contain numerous petechial hemorrhages, or are diffusely hemorrhagic. The stomach, cecum and colon may contain petechial hemorrhages, and the small intestine often is severely hemorrhagic. Small petechial hemorrhages may be seen on the serous surfaces. Hemorrhages of variable size and distribution occur in the heart.

Specific gross lesions are observed only in the lungs and liver. Gross lesions of the lungs occur in 80 to 90 per cent of infected fetuses, while lesions of the liver are present in slightly more than 50 per cent. The thorax often contains from a slight excess to two liters or more of clear serum-like fluid. The lungs and mediastinal tissues are edematous. The lungs are firm, rigid, heavy, pit from pressure, and the interlobular spaces are separated by extravasated fluid. Usually the lungs are light in color. Extravasation of fluid into the pleural cavity and edema of the lungs are less severe as the age of the fetus approaches full term.

Lesions in the liver are subcapsular foci of necrosis, varying in size from the point of visibility to 2 mm in diameter. The necrotic foci are white to grey in color and may be observed anywhere on the surfare of the liver.

Other gross lesions are edema of the submucosal tissue and connective tissue in the region of the pharynx, larynx, guttural pouches and ventral surface of the neck. Edema may be observed in the subcutaneous tissues of the inguinal and pectoral regions and in the perirenal and sublumbar tissues. The flesh of fetuses aborted from infection by the rhinopneumonitis virus is nearly always in a fresh condition, and this is of value in differential diagnosis. Fetuses that have undergone considerable autolysis prior to abortion are rarely infected by the rhinopneumonitis virus.

The tissues most suitable for microscopic diagnosis are lung, liver, and spleen, in the order named. Fixing in 10 per cent formalin and staining with hematoxylin and eosin are satisfactory for diagnostic purposes. Acidophilic intranuclear inclusion bodies may be observed in the bronchial epithelium and alveolar cells, in the hepatic cells within and bordering the foci of necrosis, and in reticulo-endothelial cells of the spleen.

Complement-fixation and virus-neutralization tests on the serum of mares are not applicable for diagnosis of abortion caused by the rhinopneumonitis virus.

As mentioned previously, the rhinopneumonitis virus causes a respiratory catarrh and febrile illness in susceptible individuals. Horses are not immunized permanently and subsequent

attacks are modified by prior immunological conditioning. This appears to be the situation with nearly all outbreaks of abortion in central Kentucky. The disease of the mare is mild and asymptomatic, but the virus invades the fetus and abortion occurs subsequent to the mare's recovery. This is evident from the fact that fully susceptible horses completely recover from uncomplicated viral infection and are immunized within two weeks after inoculation. It appears that the virus gains access to fetal tissues during a period of viremia of the mare and that subsequently the disease of the fetus is an independent process. Complement-fixing antibody titers of mares reach their highest levels within 14 days after inoculation and usually recede if the fetus is carried 30 days or longer from the time of inoculation. An increase in titer of complement-fixing antibody immediately prior to or after abortion has not been observed in mares carrying infected fetuses for an extended period.

Rhinopneumonitis of suckling and weanling foals occurs regularly during the fall and winter in central Kentucky. The disease is widely distributed in the area and occurs on a majority of farms during the same period. Serological surveys have shown a low incidence of foals positive to the complement fixation test in September. The incidence of positive reactors increases in October, reaches its highest level in November, and recedes in December. Some groups of foals escape infection during the fall and acquire it in mid-winter. The virus spreads readily and all foals in a barn usually are infected within one to three weeks. The disease in foals may be asymptomatic. Usually a serous rhinitis and fever are observed and some foals have a reduced appetite. The temperature in uncomplicated cases may reach 105 to 106 F, but usually is between 102 and 104 F. Morning temperatures may be normal or only slightly elevated, with an increase during the afternoon. Fever may persist from two to six days:

The viral phase may be followed by a mucopurulent rhinitis. Mucopurulent rhinitis may not occur in some outbreaks and in others it develops in nearly every foal. Erratic, low fevers may be observed. Coughing occurs in relatively few outbreaks. Conjunctivitis, edema of the legs, and pulmonary complications are very infrequent. Uncomplicated cases recover in 6 to 10 days and those with mucopurulent rhinitis usually recover in two to four weeks.

Serological studies indicate that mares acquire infection at the same time that the disease is present among the foals. However, few of the mares that show serological evidence of infection abort. On farms that had mares abort and the epizootiological pattern was established, abortions occurred from one to three months after the disease was present in the foals. This period of time coincides closely with the known incubation periods for abortion in mares inoculated experimentally. If 30

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to 90 days are added to the period of highest incidence of the disease among foals, we are placed in the period of January, February and March when the highest incidence of outbreaks of abortion occur. Evidence presently available indicates that the annual epizootic in foals is the principal source of virus for exposure of pregnant mares. This concept is supported by results of complement-fixation tests of all mares on affected farms at the time the first abortion occurred. The prevalence of high titers indicated that on almost every farm nearly every horse had acquired infection at some time before the first abortion occurred. The placenta, allantoic, and amnionic fluids of aborted fetuses contain virus may initiate an outbreak. Such outbreaks are observed occasionally when a newly acquired mare aborts soon after arrival on a farm. Also, it is possible for some mares to escape infection during the fall and acquire it in mid-winter from an aborted fetus.

Vaccines for control of abortion caused by the rhinopneumonitis virus have been used experimentally in central Kentucky for 15 years. The first vaccines were made from liver tissue of equine fetuses; later preparations were made from lung tissues of equine fetuses; and more recently the vaccine has been made from liver of hamsters. The virus in each vaccine was inactivated with formalin. Initially, the vaccine was administered intradermally, but after determining that a higher titer of complement-fixing antibody was obtained from subcutaneous administration the latter route was used. Results obtained from field use of the vaccines prepared from fetal tissues were equivocal. Vaccine prepared from equine tissue was discontinued because immunization against erythrocytes in the vaccine caused hemolytic disease of newborn foals.

Vaccine prepared from hamster tissue stimulates development of complement-fixing antibody titers in pregnant mares comparable to those resulting from natural infection, but the vaccine does not induce development of appreciable complement-fixing antibody in susceptible weanling foals. The titer of virus-neutralizing antibody is maintained between 10^5 and 10^8 hamster 1. d. $_{50}$ per ml of serum in pregnant mares by administering a 5 or 10 cc dose subcutaneously at 60-day intervals. Similar vaccination, however, causes very little or no development of virus-neutralizing antibody in weanlings. The efficacy of the hamster tissue vaccine for preventing abortion has not been established. Preliminary experiments indicate that the killed virus vaccine prepared from hamster tissue does not induce serviceable immunity against respiratory infection in weanlings.

VIRAL ARTERITIS

As mentioned earlier, this disease undoubtedly has been included among the disorders described as equine influenza. It is evident also that descriptions of influenza in our veterinary texts include the disease caused by the rhinopneumonitis virus,

the disease resulting from bacterial complications of viral infections, and possibly viral infections that have not been identified. After becoming acquainted with experimentally produced viral arteritis, it was possible to select from descriptions of influenza "forms" which may have been due to the arteritis virus. These forms are pinkeye, epizootic cellulitis, intestinal, a peracute illness, and possibly pulmonary. No one of these forms occurred in the experimental horses with exclusion of the others, but one or more of these manifestations may be absent in an individual. Some pulmonary forms described in the literature on equine influenza probably result from pyogenic complications involving the lungs, but respiratory distress may be observed in uncomplicated viral infection.

The symptoms presented here were observed in 39 horses inoculated experimentally. All symptoms were associated with uncomplicated viral infections, except when noted as complications. The viral inoculums were prepared in most instances from lung or spleen tissue of aborted fetuses. Other sources of virus were spleen tissue from young or adult horses and plasma obtained during the febrile period. Inoculations were made intravenously, subcutaneously, intratracheally, or intranasally. Fatal issue occurred after each route of inoculation. Thirteen of 39 horses died, all except two of uncomplicated viral infection. The disease was allowed to run its course without treatment.

Fever, leukopenia, excessive lacrimation, conjunctivitis, serous nasal efflux, and congested nasal mucosa were observed in all horses.

Fever appeared 1 to 4 days after inoculation and persisted 4 to 9 days. Maximal temperatures varied from 102 to 106 F. Temperatures usually were higher during the afternoon.

Excessive lacrimation appeared within 2 to 5 days and persisted for 3 to 15 days. In many horses the excess fluid drained through the lacrimal duct, and in others it overflowed and drained down the face. This symptom occurred in the absence of palpebral edema, but was more severe and persisted longer in horses with palpebral edema.

Congestion of the conjunctiva appeared in 3 to 6 days and persisted 3 to 15 days. The conjunctivae were slightly or deeply congested and sometimes contained petechial hemorrhages.

Palpebral edema occurred in 20 of 39 horses. It was evident in 4 to 7 days and persisted 3 to 15 days. It varied from a slight thickening of the submucosa to extensive swelling which closed the eye. In a few subjects the conjunctivae protruded past the margin of the eyelid.

Keratitis was observed in 6 horses. It appeared in 5 to 8 days, persisted for 6 to 18 days, and was associated with purulent conjunctivitis or prolonged conjunctivitis with palpebral edema.

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Photophobia was observed in 13 horses. It appeared within 3 to 8 days and persisted 3 to 15 days. Clouding if the aqueous humor occurred in some horses. It appeared as a white precipitate, first in the ventral part of the anterior chamber and then progressed until one-fourth to two-thirds of the aqueous humor was involved. The precipitate tended to remain at a horizontal level relative to the position of the horse's head. The aqueous humor in the upper portion of the anterior chamber remained clear.

Serous nasal efflux was observed in each of the 39 horses. It appeared in 2 to 5 days and persisted for 3 to 12 days. These periods were closely parallel to those of clinical rhinitis, conjunctivitis, and excessive lacrimation. The nasal mucosa was mildly to severely congested and it sometimes contained petechial hemorrhages. It became deep red and cyanotic in critically ill horses. A mucopurulent nasal exudate was observed in 6 horses, in which it appeared in 6 to 12 days and persisted for 7 to 14 days.

Pulmonary involvement was manifested by a heave line in 37 horses and dyspnea in 35. These symptoms appeared in 2 to 5 days and persisted 2 to 10 days, but usually were resolved in 3 to 7 days. Flaring of the nostrils during inspiration was frequent. The heave line was almost imperceptible in some horses, but in others it was quite pronounced. Walking or trotting affected horses 50 to 200 yards resulted in rapid respirations, flaring of the nostrils, and accentuated heave lines. In severely affected horses exercise caused cyanosis and pronounced dyspnea accompanied by almost spasmodic contraction of the thoracic and abdominal muscles. Lesions of horses infected fatally indicated that the respiratory difficulty resulted from pleural effusion, edema of the lungs, and cardiac disturbance.

Generalized weakness was observed in 31 horses. Depression and reduced sensation were observed in 33. These symptoms were evident within 2 to 7 days and persisted 2 to 12 days, but in most horses they were resolved in 6 to 8 days. The horses had a tendency to move about uneasily early in the disease. Later they were depressed, sleepy, and indifferent. Response to painful stimuli was reduced or lost. Weakness became more pronounced near the end of the febrile period and was accompanied by reeling, swaying, incoordination and a tendency to drag the toes. Horses stood with the head hanging, the ears drooped, the eyes partially closed, and the lips and tail flaccid. The anal sphincter was relaxed and in some colts the penis protruded from the sheath constantly. Some subjects developed a twitching of the nose; some intermittently clapped their lips; and others exhibited purposeless chewing movements.

Anorexia occurred in 33 horses, appearing within 2 to 6 days and persisting for 2 to 12 days. It followed soon after the

onset of fever. Feed and water were taken regularly in midly affected horses. Subjects affected moderately ate and drank erratically and horses affected severly refused feed entirely.

Colic was observed in 27 horses. It appeared in 3 to 6 days and persisted 2 to 5 days. The symptoms were variable, being almost imperceptible in some horses while others exhibited almost every recognized symptom of abdominal pain. Diarrhea was observed in 20 horses and was usually preceded or accompanied by colic. It appeared in 3 to 6 days and persisted 1 to 6 days. Diarrhea and colic were accompanied by dehydration and in some horses by severe loss of weight. Icterus was evident in 16 horses. It appeared within 5 to 7 days, persisted for 5 to 7 days, and was most pronounced in subjects with severe involvement of the intestines.

Edema of the legs was observed in 16 horses and edema of the abdomen in two. These symptoms appeared in 4 to 8 days and persisted 3 to 8 days. Edema of the legs was mild in most cases and affected only the pasterns, the fetlocks, and the metacarpal and metatarsal regions. The pelvic limbs were affected more often and more severely than the thoracic limbs. In a few subjects the legs were swollen to uniform diameter from the gaskin to the coronet.

Horses affected mildly had temperatures of 102 to 105 F for 3 to 6 days. Serous rhinitis, conjunctivitis, and occasionally edema of the legs accompanied the fever. The appetite was unaffected or only slightly impaired. Clinical recovery was complete in 5 to 8 days after inoculation. Symptoms were more pronounced and persisted longer in more severely affected horses. These usually recovered from acute symptoms in 8 to 12 days, but edema of the legs, palpebral edema, and anorexia persisted longer in some horses.

Acutely affected horses developed temperatures of 104 to 106 F, colic, diarrhea, respiratory difficulty, severe depression, and weakness in 3 to 6 days, or they were prostrate or died in 5 to 8 days. Clinical recovery usually occurred in 10 to 14 days. Fatal issue was associated with early and persistent colic and diarrhea, muscular weakness, accelerated respiratory and cardiac rates, and rapid prostration. The body temperature often fell to normal or subnormal 6 to 24 hours before death. The disease was more severe in pregnant than in barren mares, more severe in aged mares and weanlings than in young adults, and more severe in poorly nourished, poorly fleshed, heavily parasitized horses than in well nourished individuals that were relatively free of internal parasites.

Thirteen of 39 horses infected experimentally with the arteritis virus died. Eleven deaths were due to uncomplicated viral infection. One foal that died on the sixth day had a strep-

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tococcic septicemia which apparently developed during the agonal period. A mare that died on the thirteenth day had a few small areas of red hepatization in the apex and cardiac notches of the lungs. Seven of the horses infected fatally were weanlings 5 to 6 months old. Three died on the sixth day, one in the seventh, one on the eighth, one on the ninth, and one on the ten day after inoculation. Five pregnant mares that died were between 5 and 8 years old and the deaths occurred on the fith, sixth, seventh, eighth, and thirteenth days.

The conjunctival were edematous, congested, and contained petechial hemorrhages. The mucosal surfaces of the upper respiratory tract were congested and contained few or numerous petechial hemorrhages. Edematous infiltration of the subcutis and excessive fluid in the joints and tendon sheaths were observed in horses with affected legs.

The pleural cavity contained from a slight excess to 10 liters of serum-like fliud which often contained strands of fibrin. Blood vessels of the costal pleura were injected. The mediastinal tissues and the adipose tissue of the heart and pericardium were edematous and occasionally contained petechial hemorrhages. The surface of the lungs was smooth, but irregular due to emphysema, which was most severe in the apex, cardiac notch, and diaphragmatic border. Edema of the lungs was not severe. Usually it was evident by increased weight, by a translucense of the pleura, by separation of interlobular spaces in the apex, cardiac notch, and diaphragmatic borders, and by dilation of the subpleural lymph vessels. Petechial hemorrhages were observed under the pleura. The mucosa of the major bronchi was congested and contained petechial hemorrhages. The mediastinal lymph nodes were edematous and slightly enlarged.

The pericardial sac occasionally was distended with fluid. Hemorrhages were observed on the epicardium and endocardium. The heart muscle was pale and sometimes yellowish.

A serum-like fluid containing strands of fibrin was found in the peritoneal cavity of most horses. The quantity varied from a slight excess to 8 to 10 liters. Petechial hemorrhages were frequent in the omentum and peritoneum. The adipose tissue in the sublumbar and perirenal regions, around the origin of the anterior mesenteric artery, and along the course of the cecal and colic blood vessels was edematous in nearly all horses. The abdominal lymph nodes were edematous, slightly enlarged, and occasionally hemorrhagic. The kidneys were enlarged and usually had a pale cortex and congested medulla. The adrenals were enlarged, and the cortex was diffusely hemorrhagic or contained discrete hemorrhages.

The broad ligaments and uterus were edematous in some pergnant mares. This lesion was present in mares that did not

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abort and in those that aborted prior to death. Edema of the uterus was both subserosal and submucosal. The pancreas was edematous in some horses. Multiple infarcts were found in some spleens. Spleens of two horses were greatly enlarged and engorged with blood. The liver had no characteristic lesions.

The gastric mucosa was congested and contained petchial hemorrhages. Hemorrhages were frequent in the mucosal surface of the small intestine. The mucosa of the cecum and colon often contained areas of hemorrhagic and diphtheritic inflammation. Submucosal edema affecting the small intestine, cecum, and colon was the most striking lesion. Alternate pirtions of the small intestine were slightly to severely edematous, with thickness of the wall varying from 8 to 15 mm. Edema of the cecum was most severe at the apex, reaching a thickness of 1 to 2 cm. The dorsal colon was affected less severely than the ventral colon, in which thickness of the wall varied from a slight increase to 2 cm. Subserosal edema occurred along the course of the large blood vessels of the cecum and colon.

Horses dying from peracute disease had no lesions except minute petechial hemorrhages on the mucosal and serosal surfaces.

Abortion is likely to be the most costly effort of arteritis of pregnant mares. In three outbreaks, the incidence of abortion varied from 50 to 70 per cent.

Mares inoculated experimentally aborted between the seventh and thirteenth days. In natural outbreaks, mares usually aborted during the latter part of the period of acute symptoms or during the convalescent period. Some mares aborted without illness being detected by the stable attendants. Abortion occurs without premonitory symptoms. The placenta often is expelled with the fetus. The foot pads and inner surface of the amnion may be stained yellow.

Aborted fetuses do not have lesions which permit specific diagnosis of infection by the arteritis virus. Gross lesions are similar to some of those resulting from infection by the rhinopneumonitis virus, but there are distinguishing lesions and features. Severe edema of the lungs and focal necrosis of the liver differentiate infection by the rhinopneumonitis virus. Histologically, intranuclear inclusion bodies in the lungs, liver, and lymphoid tissues are specific for infection by the rhinopneumonitis virus. The arteritis virus does not induce development of inclusion bodies. Outbreaks of abortion caused by the rhinopneumonitis virus are not associated with evident illness of the mares. Abortions due to infection by the arteritis virus have occurred concurrently with obvious illness in every outbreak identified. Hemorrhages occur in the mucous membrances of the respiratory and alimentary systems of fetuses infected by

either virus, but are more pronounced in those infected by the rhinopneumonitis virus. Edema of the pectoral, inguinal, and sublumar tissues is present more regularly and is more severe in fetuses infected by the arteritis virus. The lungs of fetuses infected by the arteritis virus may be slightly edematous, but normal color and elasticity is retained. With rhinopneumonitis, the lungs are severely edematous as noted previously. Pleural effusion is less frequent and less severe in arteritis of the fetus. Excessive fluid in the peritoneal cavity is more frequent in fetuses aborted from infection by the arteritis virus. There are no characteristic gross lesions in the spleen in fetal rhinopneumonitis, but with arteritis single or multiple in farcts may occur. Fetuses infected by the arteritis virus often die *in utero* and are severely autolysed when expelled, whereas those having rhinopneumonitis virus infection are seldom autolysed.

There is little information on current prevalence and distribution of equine arteritis. The virus has been isolated and identified by cross protection tests from outbreaks in Ohio, Pennsylvania, California, and Kentucky. Unconfirmed diagnosis indicate that the disease has occurred at widely separated points throughout the midwest. All outbreaks identified have been in Standardbred horses. The virus has not been propagated in laboratory hosts. No vaccine is available. The virus is not affected by penicillin or streptomycin. The duration of immunity following recevery from the disease has not been determined.

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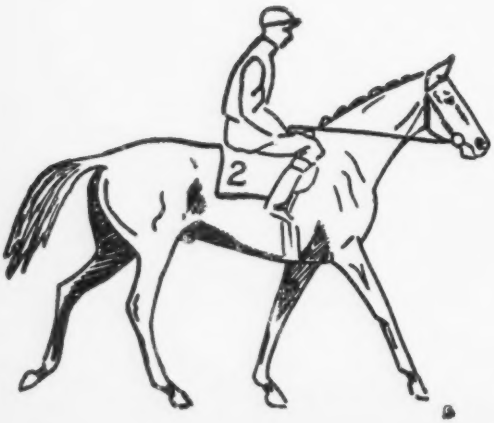

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